

nutans and laryngismus are so rare that my previous admission will be pardoned. Likewise the words of Drs. Adams and Blackraeder are not irrelevant. The former says: "There may be a few, like myself, that have never seen laryngismus stridulus complicating rickets." The latter says: "In Montreal I may say that the cases of laryngismus complicating rickets are comparatively few in English hospitals, and a very infrequent symptom in this country."

In Europe, this disease associated with tetany occurs isolatedly and in epidemics and epidemics. Gangenhöfer, Escherich and Loos, call tetany an infectious disease; possibly the other local spasms under discussion belong to the same category. Clinical facts indicate protean etiologic derivatives, rather than any one or few causative agencies for torticollis, spasmus nutans and laryngismus stridulus.

The dissentient views of able teachers is in part responsible for this confusion and uncertainty. Escherich, Loos, Kassowitz, Gangenhöfer, Gay and Soltman are a few of those giving earnest attention to the relation of season, age, heredity, unhygienic surroundings, infection, etc., to laryngismus and tetany. The two first named pronounce laryngismus but a symptom of tetany that does not stand in any causal relation to rachitis. This brings a storm of protest from Kassowitz who is the declared exponent of the rachitic genesis of laryngismus.

The craniotablic theory of Elässer does not seem tenable. Nor are Kassowitz's pathologic findings in the skull-cap, membranes and cerebral lesions in rickets, proof positive of their kinship to the dangerous respiratory symptoms of laryngismus.

"The latter," says Gay, "does not necessarily fall upon those who have the most marked signs of rickets." Again, laryngismus is present where rickets can not be demonstrated.

On the other hand, laryngismus is wanting in cases of most marked rachitic deformity where excessive weight and size of the head, and muscular weakness, or irregular and diminished thoracic capacity might lead to sudden serious respiratory disturbance.

Thus, we see, the question is yet an open one. Escherich and Loos, in their study of the relationship of tetany to laryngismus, are clearing the field for a broader and more impartial view of the entire subject. Their work is supported by the clinical investigations of Gangenhöfer, Hoffman, Neusser and Jasch, Gay, Abercrombie, and Smith, who describe cases of tetany either associated with laryngismus, or of laryngismus alone in which contractures of the extremities are present, but which is marked by the three cardinal signs of tetany, viz.: Trousseau's phenomenon, the facial phenomenon, and general neuromuscular hyperkinesis.

Gay, in dwelling upon the importance of the facial irritability says: "It is not limited to the seventh nerve, but is significant of an increased excitability, probably of all the motor nerves of the body." "It seems to bridge over and connect laryngismus with tetany as it occurs in children."

To sum up:

1. Torticollis, spasmus nutans, laryngismus stridulus, and possibly tetany are closely allied neuroses, consequent upon their common anatomico-physiologic characteristics.

2. Their relation to commonly accepted causes, such as heredity, environment, rickets, etc., is admitted.

3. Their etiology is undetermined.

4. They may be of an infectious origin and, as it were, the common, the comparative and superlative degrees of one and the same affection.

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## CAUSATION AND EARLY TREATMENT OF MENTAL DISEASE IN CHILDREN.

Read in the Section on Neurology and Medical Jurisprudence, at the Forty-fifth Annual Meeting of the American Medical Association, held at San Francisco, June 5-8, 1894.

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When I state that this paper is intended to deal with the physical causes which underlie the mental defects of the young, it may appear to you that it could be more fittingly read before specialists in their care and treatment. I hope to demonstrate to you, however, that a very large responsibility antedates their admission into institutions for the feeble-minded, and rests on no one as heavily as on the family physician. It has long been held that mental enfeeblement in children, in the vast majority of cases, was purely non-development of the nervous centers; and this view has been quietly accepted by the general profession and the public at large. Many a family with a clean history of mental health, when a bright child has suddenly changed and mental growth has ceased, perhaps after some slight illness, or perhaps after no noticeable physical dis-

turbance, can not view the misfortune in the same way as they would look on an injury to a limb or any bodily organ; but feel it as a reproach.

Year by year, as opportunities for studying the clinic and pathologic history of these unfortunates increase, it is more apparent how largely their infirmity results, as the residual effect of acute disease. It is to such cases; the diseases which most frequently leave such lasting and serious effects, and the brain changes they induce—with some suggestions regarding their early treatment, that I ask your attention for a few minutes.

The various forms of agenesis, though interesting, we may pass without comment. The laws of heredity are entirely beyond our control; and the marriage of the unfit, with offspring certainly destined to become a burden, is scarcely less so under our present social conditions. The census of 1890 showed over 95,000 feeble-minded in our country, and their number at present will approximate and probably pass the 100,000 mark. This great number may be roughly divided into cases of primary imperfect development, and into what may be looked upon as a juvenile dementia, the result of destructive change in the brain tissue. The development of the brain may be retarded by disease in early years. The growth of the organ in the first years of life is exceedingly rapid. Tuczek, basing his statements on investigations by Huschke and Bischoff, estimates the daily increase in the size of the brain, during the first year of life, as more than one cubic centimeter. Dr. Robert Boyd estimates the brain weight at birth, ten ounces to eleven and one-half ounces. In thirty-four children between one year and two years, it averaged thirty-two and twenty-five one-hundredths ounces. Between two years and four years thirty-eight and seventy-one one-hundredths ounces. It will be readily understood that any interference with nutrition in this period, if continued for some weeks, as it might be by any disease, would be apt to leave its permanent mark on the growth and full development of the organ. This theory is borne out in fact. In seventy-five brains taken from all classes of feeble-minded children, I found the average weight only thirty-eight and three-tenths ounces. It is rare to find their brain above forty-five ounces in weight, and when it is so found it is generally in cases where the mental defect developed in the later years of childhood; in children when the mental trouble is very slight, or in low grade cases with cerebral hypertrophy. In only eight of my cases was the patient under 10 years old. Such cases are not to be classed with cases of congenital non-development, which, being an inherent fault in the brain, can not be combated as can the condition just described. With congenital or prenatal arrested development, we may class victims of convulsive attacks, which appear before the beginning of dentition. Also cases of injury from prolonged labor or forceps pressure; and they are by no means few. No intelligent physician would apply injudicious pressure on the thin skull and soft brain of the infant except to avoid still greater danger; on the careless or unscrupulous operator all warnings are wasted. It would be unwise to pass over all forms of imperfect development as beyond the reach of medical skill, without some allusion to craniectomy for the relief of microcephalus from premature ossification of the bones of the skull. This operation has, of late, been performed several

times; and from a surgical point of view, successfully. From my own observations, I am strongly of the opinion that the small skull is the *result* of the small brain rather than the *cause*. Evidence of pressure I have never seen except in one case. The true value of the operation can only be determined when a sufficient number of operations have been performed to make an intelligent comparison with cases who owe their improvement to school training alone, which is often very successful in such cases, unless sclerosis accompanies, (or perhaps causes) the retarded growth.

With all these omissions, the number of cases where brain lesion and consequent mental enfeeblement appear and originate in easily recognizable disease, at an age when medical treatment may be intelligently pursued, will be found to be very large.

Through the courtesy of authorities at the Elwyn School for the Feeble-Minded, I have been able to examine 1,000 histories from their records, choosing those series of cases which contained the most cases under my own care during my eight years residence in that institution; which, by knowledge of the children and their friends, I had excellent opportunity for verifying.

I have omitted all cases where direct heredity could be traced, when the infirmity appeared to be congenital or the result of accident at birth, or where spasms occurred or lack of ordinary intelligence was noticed before the age of 6 months, and when the patient was too young to decide as to his intelligence at the onset of the alleged causative disease; regarding such cases as possibly congenital. I have declined to count any case said to be due to traumatism, unless spasms, paralysis, or other symptoms of nervous shock directly followed the accident.

Notwithstanding this careful pruning, no less than 322 cases out of the 1,000, appear to have been the direct result of disease which would ordinarily need and receive the physician's care. Quoting figures like these at the International Conference of Charities at Chicago last summer, a foreign representative asked: "What are the doctors doing in these cases?" A question I did not attempt to answer.

The following table will show the relative frequency of the diseases in which the cerebral mischief appears to originate, and the age of onset:

	6 months.	6 to 12 months.	12 to 18 months.	18 to 24 months.	2 to 5 years.	5 to 10 years.	10 to 15 years.	Unknown. <sup>1</sup>	Total.
Spasms of dentition . . . . .	2	20	25	19					75
Traumatism . . . . .	3	10	2	4	20	6	3	2	51
Cerebral inflammations . . . . .	3	3	3	3	14	2	4	3	42
Scarlet fever . . . . .	3	2	3	3	15	9	1	7	42
Epilepsy of unknown origin . . . . .					10	3	5	2	20
Mental shock (fright) . . . . .		1	1	1	4	2	2	1	11
Gastric and typhoid fever . . . . .		1	1	1	5	1	1		9
Whooping cough . . . . .	1				2	1			9
Measles . . . . .		2	2	1	2	1		1	7
Exposure to heat (sunstroke) . . . . .			1		3	3	1		13
"Fever" (form unknown) . . . . .	1			1	8	3			16
Cholera infantum . . . . .		1	4			2		1	5
Smallpox . . . . .		1	1						4
Malarial fever . . . . .		2	1			1			3
Infantile paralysis . . . . .	1	1	1						3
Diphtheria . . . . .	1	1	1						3
Vaccinia . . . . .		1	1	1	1				3
Marasmus . . . . .	1								2
Catarrhal fever . . . . .		2							2
Abscesses . . . . .			1						2
Exposure . . . . .					1				1
Erysipelas . . . . .					1				1
Poison (Wild Lilac) . . . . .					1				1
Self abuse . . . . .							1		1

<sup>1</sup> While the age is not given, in these cases, the history shows that the child was beyond early infancy.

It will be seen that convulsions, occurring within the period of dentition, heads the list. Then comes traumatism, which includes all injuries from blows or falls on the head. Among the specific fevers, scarlet fever takes the lead for its destructive effects on the nervous system; although the cerebral inflammations claim an equal number. Fright occasions an unexpectedly high number, but in each case counted, the history seemed completely to substantiate this as the active cause.

Let us compare with this table the result of 300 autopsies collected from many sources:

Hemisphere diseased.	Right.	Left.	Both.	Not stated.	Totals.
Atrophic sclerosis . . . . .	21	14	38	23	96
Porencephalus . . . . .	14	9	15	9	47
Porencephalus and atrophy . . . . .	3	6	4	1	14
Agensis . . . . .	1	6	9	6	22
Tuberous sclerosis . . . . .	1	1	11	0	13
Atrophy with internal hydrocephalus . . . . .	0	0	2	0	2
Atrophy with cyst . . . . .	1	0	1	0	2
Atrophy with hypertrophic skull . . . . .	0	1	2	0	3
Hydrocephalus . . . . .	0	0	17	0	17
Thickened membranes . . . . .	0	0	14	0	14
Thickened membranes and vessels . . . . .	0	0	2	0	2
Defective corpus callosum . . . . .	0	0	29	0	29
Microcephalus . . . . .	0	0	10	0	10
Hypertrophy . . . . .	0	0	15	0	15
Hypertrophy with sclerosis . . . . .	0	0	1	0	1
Cyst . . . . .	3	0	0	2	5
Primary disease in cells, fibers, or both . . . . .	2	0	3	3	8

It will be seen at a glance how relatively few are the cases, when conditions of non-development alone are the dominant defect in this table. It may be justly said that in such a large number, collected from every possible source, many of them intended to illustrate the cause of *physical* defect rather than *mental*, that the true proportion may not be here represented. I beg leave therefore to quote from 100 consecutive autopsies made at the Elwyn institution, in which in 54 per cent. conditions were found constituting the residual effects of former disease or traumatism. The initial stage of some of these, no doubt, occurred so early in life that their nature, and possibly even their existence, might not be determined. Many others probably occurred at an age when there was good opportunity for careful study and perhaps helpful treatment.

Glancing at the table, the large number of cases of sclerosis and porencephalus will be at once noticed. These are both terminal conditions; and when fully established, it is doubtful if they can be relieved by any means within our present knowledge. Closely allied to these changes, we find the thickened and adherent membranes which follow meningeal inflammation, and which must necessarily interfere with the large blood supply so essential to the perfect functional life of the cerebral cortex. Of their early medical treatment we will speak a little later.

An interesting disease, fortunately quite rare, is tuberous sclerosis (*sciérose tubéreuse* of the French writers). From a study of the histories of these cases, which I have been able to verify by autopsy, I should describe its clinical history as follows: Its onset is sudden, and demonstrates itself by spasms of only moderate severity, but nearly continuous. They last always several hours, and sometimes for days, and are generally localized in certain groups of muscles, rather than extending to general spasm. The rise in temperature is moderate. Stupor is continuous but may not be profound. The spasms are intractable under every form of treatment I have

ever tried, and only ceased when the areas involved are functionally dead. If the child survives, it will be found that certain cerebral functions, corresponding to the areas attacked, are entirely and permanently obliterated. In no form of brain disease with which I am acquainted is the loss so sudden, complete and lasting as in this form. Post-mortem, we find rarely single, generally several areas in the cortex, quite sharply defined to the eye, gray white in color and slightly elevated. Microscopically it appears to be due to a finely granular effused material, which presses on the normal elements of the cortex, obliterating the lymph spaces. The functional activity of the cells is first destroyed from pressure, and they soon atrophy. A little boy at Elwyn was found to have several areas of this disease, but his mother insisted that he had never had either a fit or any severe illness. That certain faculties (articulate speech for one) had never shown the slightest tendency to develop. This has led me to suspect that this condition might form in utero, as his mother was subject to spasms during her pregnancy. Though only eight years old, he had a well formed brain weighing forty-eight and one-quarter ounces. The diagnosis of this disease is not difficult, its prognosis is distressingly easy, and I know of nothing which can greatly relieve it. I have dwelt on it to this length, because it is not generally mentioned in text-books, and it is desirable to eliminate in diagnosis of more tractable affections.

From our examination of the pathologic conditions underlying mental enfeeblement, it is evident that medical treatment, at the time they are placed in institutions, is liable to arrive too late to be of much service. By the time the child has reached the age of seven years, the brain has finished its period of most active growth. The hypertrophied brain has its bulk of interstitial tissue usurping the space and nourishment needed by the true brain cells and fibers for their own proper development; and the brain which has suffered from violence or destructive inflammatory process, has its mass of sclerotic scar tissue, whose influence on the healthy portion of the organ is that of a local irritant, tending to induce spasm or an increasing area of disease. However refractory such cases may be at their maturity, it is by no means true that they are equally so at the beginning. Let us take up some of the clinical causes of mental enfeeblement in the order of their frequency.

First among them is spasm occurring during the period of dentition. We find no less than 72 of our 322 cases have no other assignable cause for the subsequent mental decadence. Although in a large number of cases the convulsions ultimately cease, mental development is found to have been interfered with. All infantile disease which places any special strain on the system during this critical period, is liable to cause the same danger, and it will be generally found that the brains of the imbecile weigh less than those of normal children, and we have acquired imperfect development as a complication. The spasms of dentition is a subject deserving the most careful study and treatment.

It is a grave mistake to forget that the spasm by itself is but a symptom. That, however alarming it may be to the friends, it rarely does permanent harm. It is not scientific practice in such cases to numb the brain with large doses of bromid, and take

no means of strengthening its inhibitory power by exercising every known means of building up the nervous system. Plenty of fresh air is an essential. Moderate exercise, tonics, especially the phosphates, long continued. Delay all attempts at school education until assured of recovery, and above all other things guard the child's diet. It is my opinion, formed by years of study of large numbers of epileptics, that there is no more potent factor in the production of spasm in children than irritation of the gastro-intestinal tract. In a recent study of 566 cases of juvenile convulsion, in which the histories appeared full and reliable, in 52½ per cent. the convulsions had ceased although mental enfeeblement had in some degree persisted. I believe as many would have recovered without so many cases of mental impairment, had the cause shared the treatment with the symptom.

Next in order comes traumatism, including blows, falls and all other injuries to the head, from which I have excluded all cases which were not followed by spasms, paralysis or other symptoms of severe nervous shock. It is comparatively rare for operative procedure to be made in these cases, partly, no doubt, because friends will not allow them. It is a very doubtful matter how much good operation would do, except where fracture occurs; for post mortem examination demonstrates that the injury from blows is apt to be diffuse rather than local. Complete rest for some time after the accident should be insisted upon, and the appearance of a spasm after the primary irritation has had time to subside, should awaken the gravest apprehension. Secondary mischief has almost certainly been awakened and such cases are most obstinate. The treatment recommended for early convulsions should be begun as early as possible. I would not discourage operative measures for such cases in these days when such operations can be done with relative safety.

It is difficult to believe that large doses of bromids should accomplish anything in these cases, except to smother the spasms and blind both physician and friends as to the progress of the disease. Of the medical treatment I will speak later. The specific fevers are seen to be a very fertile cause of subsequent cerebral mischief, especially scarlet fever. It is not at all uncommon for convulsions to occur at some period of its course. In measles, if the nervous system is weak, meningeal symptoms are very apt to complicate the case. At the Pennsylvania Institute for Feeble-Minded Children, three epidemics of measles occurred, covering 397 cases. In the school department, when the children were of more healthy growth, catarrhal complications predominated; but in the asylum department, cerebral complications were the rule in the many cases where complications occurred, in some cases so severe as to kill the child in two or three days. When pneumonia would fatally attack a measles case, meningeal congestion would also be found post-mortem. In all such cases, as soon as indications of intercranial irritation are seen, prompt derivative measures should be employed to relieve cerebral congestion before the foundation be laid for permanent changes. Bromids may enter freely in the treatment at this stage but should not be long continued. The general remedies for this purpose are too well known to need mention. The number of imbeciles left in the wake of acute inflammatory cerebral disease is well known. Only

the comparative infrequency of this class of affections prevents this number from being frightful.

In all these diseases which I have described it is the residual products which cause the dreaded after effects. The remedies which are known to be of service are not numerous. Among those on which I have learned to look with favor is chlorid of gold. My use of it has been limited, but occasionally it has proved very effective. Much more can I commend a combination of iodid of potash with iodid of iron. In children where there has seemed to be a reawakening of cerebral change, and extension of old trouble has threatened, the symptoms have abated and the children have gained flesh and strength under its use. Arsenic, though less certain, has occasionally done good service.

The most satisfactory treatment is for the physician to follow his case through convalescence to complete health. To advise regular habits, sufficient rest, and above all things, and before all things, to provide rest and food for the rapidly growing brain, whose needs in the very young dominate all other organs, and the retardation of whose growth may be lifelong.

Lastly, I will refer to those obscure cases of epilepsy which arise in the years between dentition and the advent of puberty without any cause that we can demonstrate, merely to confess my inability to understand their origin or to recommend special method of treatment. They will occasionally recover under almost any treatment. Bromids will best diminish the frequency of their spasms, and, I believe, shortens the period during which they are fated to suffer.

## THE FUNCTIONS AND THE FORM OF THE OBSTETRIC FORCEPS.

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The functions of the obstetric forceps may be divided into constant, occasional, extraordinary and incidental. Its sole constant function is traction—the reënforcement of the natural *vis-a-tergo* by an artificial *vis-a-fronte*. As traction is intended to supplement the natural forces of labor, it should imitate them in so far as an artificial process may imitate a natural one—it should be intermittent, it should be moderate, it should urge the presenting part by its smallest circumference along the line of least resistance which in a general way will coincide with the axis of the parturient canal, at whatever point the presenting part may be engaged, and as nearly as may be with the axis of the blades of the forceps. Minor exceptions, however, are not infrequent. When the head impinges too strongly on the pelvic walls, either laterally or anteriorly, the line of least resistance will traverse the pelvic axis towards the opposite wall. When the forceps is applied before rotation takes place, the axis of the blades will depart from the axis of the pelvis proportionately to the obliquity of the application; at the outlet the interests of the perineum require that traction be anterior to this axis. Traction by the ordinary forceps as usually employed is neither moderate nor in the line of least resistance, and it is likewise neither in the axis of the blades nor in that of the pelvic